Adaptive remodeling at the pedicle due to pars fracture: a finite element analysis study

Laboratory investigation

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Object. Spondylolysis is a common condition among the general population and a major cause of back pain in young athletes. This condition can be difficult to detect with plain radiography and has been reported to lead to contralateral pars fracture or pedicle fracture in the terminal stages. Interestingly, some patients with late-stage spondylolysis are observed to have radiographic or CT evidence of a sclerotic pedicle on the side contralateral to the spondylolysis. Although computational studies have shown stress elevation in the contralateral pedicle after a pars fracture, it is not known if these changes would cause sclerotic changes in the contralateral pedicle. The objective of this study was to investigate the adaptive remodeling process at the pedicle due to a contralateral spondylolysis using finite element analysis.

Methods. A multiscale finite element model of a vertebra was obtained by combining a continuum model of the posterior elements with a voxel-based pedicle section. Extension loading conditions were applied with or without a fracture at the contralateral pars to analyze the stresses in the contralateral pedicle. A remodeling algorithm was used to simulate and assess density changes in the contralateral pedicle.

Results. The remodeling algorithm demonstrated an increase in bone formation around the perimeter of the contralateral pedicle with some localized loss of mass in the region of cancellous bone.

Conclusions. The authors’ results indicated that a pars fracture results in sclerotic changes in the contralateral pedicle. Such a remodeling process could increase overall bone mass. However, focal bone loss in the region of the cancellous bone of the pedicle might predispose the pedicle to microfractures. This phenomenon explains, at least in part, the origin of pedicle stress fractures in the sclerotic contralateral pedicles of patients with unilateral spondylolysis.

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Key words: pedicle • remodeling • sclerosis • finite element analysis • spondylolysis • pars fracture

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Abbreviations used in this paper: FE = finite element; SED = strain energy density.
sclerosis in the contralateral pedicle. The hypothesis of this study was that the increase in stress at the contralateral pedicle after pars fracture would trigger sclerotic remodeling changes in the pedicle. Secondly, we sought to unveil the mechanism of contralateral pedicle fracture in affected sclerotic pedicles.

Methods

Multiscale Vertebral Model

A 3D FE model of the human L-3 vertebra, generated from CT images, and a high-resolution FE model of the pedicle of the L-3 vertebra, generated using micro-CT images, were combined to form a multiscale vertebral model (Fig. 1). The details of the model were described previously. The deformable elements were assigned linear material properties simulating bone (modulus of elasticity of 3000 MPa and Poisson’s ratio of 0.3). Uniform distributed loads were applied at the superior and inferior facets as well as at the superior edges of the neural arch of the vertebra to represent a hyperextension loading. The magnitude of the load was adjusted to obtain similar stress levels at the pars interarticularis and pedicle as observed by Sairyo et al. The vertebral body elements and the rigid elements of the right pedicle were assigned rigid body constraints, and fixed boundary conditions were set. Following intact testing, the elements representing the pars were removed to simulate a spondylolytic fracture (that is, a pars fracture) on the contralateral side.

Adaptive Remodeling

An adaptive remodeling algorithm is used to determine the remodeling response of bone to an external stimulus based on an empirical density-stimulus relationship. The stimulus is usually a change in the external loading, which does not cause any reaction in bone as it remains within the physiological range. If the stimulus is above or below the physiological levels (that is, homeostasis), then the resulting change in bone density in response to the stimulus is calculated. If this new density distribution is not adequate to reach a new physiological balance neutralizing the effects of the stimulus, then further changes are calculated in an iterative fashion until no change in density is required.

To enable the detection of adaptive changes at the contralateral pedicle after the unilateral pars fracture, the following adaptive remodeling algorithm was used:

$$\Delta \rho = \begin{cases} A[S - (1 \pm s)S_{ref}], & S \geq (1 + s)S_{ref} \text{ or } S \leq (1 - s)S_{ref} \\ 0, & \text{otherwise} \end{cases}$$

This algorithm dictates that if the stimulus is above \((1 + s)S_{ref}\) or below \((1 - s)S_{ref}\), then the resulting change in density will be \(A[S - (1 \pm s)S_{ref}]\). This has been commonly used in previously performed remodeling studies, where \(\rho\) denotes the apparent bone density, \(S\) and \(S_{ref}\) denote current and reference remodeling stimulus, \(s\) denotes the “lazy (dead) zone” constant (that is, the constant that determines range of stimulus that does not cause a change in density, a physiological balance), and \(A\) denotes the remodeling function coefficient. \(A\) is a function of the surface area per unit volume and time that density changes take place. The stimulus is the strain energy density (SED) per unit apparent density (that is, \(U/\rho\), where \(U\) is the SED) for each element. The reference remodeling stimulus was calculated from the SEDs from the intact vertebral model. The density distribution among the elements of the pedicle was initially assumed to be homogeneous and was calculated to be 1.014 g/cm\(^3\) using the relationship \(E = 2875\rho\), where \(E\) (modulus of elasticity) was 3000 MPa, as mentioned above; \(s\) was set to 0.4.

The initial SEDs were obtained at the aforementioned loading and boundary conditions via FE analysis. The results were then imported into a mathematical analysis software program (MATLAB, Mathworks). Increment in apparent density of each element was calculated on the basis of the stimulus value. The material property of each element was updated according to the new density values. Then, the FE analysis was run again. The density of an element was allowed to change between a maximum of 1.92 g/cm\(^3\), the density of cortical bone, and minimum of 0.001 g/cm\(^3\). The iteration was set to end when all density changes were smaller than 0.001 g/cm\(^3\), which was deemed as no change. The convergence of the solution was also monitored by using an objective function of \(F\):

$$F = \frac{1}{n} \sum_{i=1}^{n} |S_i - (1 \pm s)S_{ref}|$$

The changes of density increment and \(F\) were monitored graphically during the analysis. If the rate of the change in densities and function \(F\) markedly slowed down or reached a plateau, the iteration was stopped manually.
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Various values (0.01, 0.1, 0.2, 1, and 10) were assigned to \( A \) to observe its influence on the analysis.

**Results**

*Intact and Fractured Vertebrae*

Stress and SED distributions for the posterior vertebral body and the deformable layer of the voxel-based pedicle in intact and pars fracture cases were reported previously.\(^7\)

**Remodeling**

The maximum von Mises stress was 162.86 MPa for the voxel-based pedicle (Fig. 2). Stress at the pedicle changed in both magnitude and distribution compared with the intact and fractured states. The maximum stress occurred at the rostral edge of the contralateral pedicle in the fractured state. Those at the caudal edge, however, were also higher in both the intact and fractured groups.

There was an overall decrease in SED after the remodeling simulation (Fig. 3). High SED concentrations were localized at the perimeters of both the rostral and caudal edges of the pedicle. Despite the overall decrease in the SED at the pedicle, the highest SED value at the edges (0.91 J/mm\(^3\)) was larger than those of both intact and fractured groups.

After the remodeling simulations, the density distribution dramatically changed compared with the initial homogeneous state (1.014 g/cm\(^3\)). There was a marked increase in density around the perimeter of the pedicle. The outer margins of the pedicle shown in dark brown in Fig. 4 reached the maximum density value (1.92 g/cm\(^3\)), indicating cortical bone formation. However, the remodeling process resulted in a loss of mass in some regions of the pedicle, specifically in the cancellous bone of the pedicle. The loss of structural integrity in this region outweighed the increase in density in the outer regions, associated with pedicle cortical sclerosis. The minimum density values were 0.77 and 0.35 g/cm\(^3\) for \( A \) of 0.01 and 0.1, respectively, while for other \( A \) values minimum density was 0.001 g/cm\(^3\). The density changes resembled the stress and SED distribution at the pedicle after the fracture of the contralateral pars interarticularis.

**Coefficient of \( A \)**

Increasing values for \( A \) caused more rigorous change in the distribution. Characteristics of density increment seemed to be similar for all values of \( A \); however, those of the decrements showed greater changes as \( A \) increased; that is, the density loss was more pronounced with larger \( A \) values. For the largest value of \( A \) (that is, 10), the density of each voxel quickly reached maximum and minimum values, resulting in an unrealistic density distribution. The small values of \( A \) (0.01, 0.1, and 0.2) caused similar remodeling results. The objective function of \( F \) reflected this similarity by asymptotically converging at a similar minimum for these 3 values (Fig. 5).

**Discussion**

In this study, the adaptive remodeling analysis showed that changes in the loading of the pedicle due to a fracture of the contralateral pars caused dramatic changes in bone mass distribution in the pedicle. Higher stress regions, that is, the perimeter of the pedicle, seemed to acquire higher bone density with remodeling, while the cancellous marrow of the pedicle with lower stress showed diminished bone structure. This finding is consistent with the clinical presentation of the condition. Clinical reports suggest that patients with a terminal stage unilateral pars interarticularis fracture may develop a sclerotic pedicle, as seen on plain radiography.\(^6,17,20,21\)

Our results demonstrated that the central marrow (cancellous bone) region of the pedicle underwent substantial bone loss. This theoretically would be associated with a localized loss in pedicle strength. We speculate that this local loss of strength may be associated with a structural vulnerability of the contralateral pedicle against mi-
crofracture initiation under repetitive or sudden traumatic loading. This provides a plausible explanation for the observation of fracture of a sclerotic pedicle in patients with a contralateral pars interarticularis fracture.

If not treated, a unilateral spondylolysis may be associated with a contralateral stress fracture in the terminal stage. Contralateral fractures (pars interarticularis or pedicle fracture) may result in spondylolisthesis. Our previous study demonstrated that a unilateral pars fracture increased the stress in the contralateral pedicle and pars. It is probable that when stresses applied to the contralateral pedicle are sustained, bone remodeling and sclerotic reactions will be observed. On the other hand, when the stress levels exceed a critical level, the pace of remodeling might become insufficient to compensate for high strength demand and result in a fracture at the pedicle. However, it is not clear why sclerotic changes are observed only in the pedicle, since the contralateral pars

**Fig. 3.** Strain energy density (SED or SENER) distribution at the deformable layer of the pedicle before (left) and after remodeling (right).

**Fig. 4.** Density distribution at the deformable layer of the pedicle after remodeling iterations for various values of remodeling function coefficient.
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interarticularis is also exposed to increased stress. We speculate that the marrow-rich and vascular trabecular component of the pedicle hosting active bone cells promote the cortical ossification (sclerosis) process.

Sclerotic pedicles, although uncommon, have been reported to occur for a variety of reasons.\(^4\),\(^10\),\(^21\),\(^22\),\(^25\) Despite the diversity of the causes, the reactive sclerosis is triggered by a stress-changing event in all cases. No doubt, pedicle sclerosis and/or fracture due to unilateral spondylolysis is the most critical condition among all these clinical scenarios. Sherman et al. published one of the early reports on reactive sclerosis in the presence of a pars fracture.\(^20\) The authors highlighted the importance of probing the etiology of the sclerotic lesions. Osteoid osteoma is associated with similar radiographic characteristics. Pedicle excision without fusion has been used as a preferred strategy for osteoid osteoma. In the presence of a pars interarticularis fracture, which might not be evident on plain radiography, the removal of the contralateral pedicle would further accentuate the instability. This underscores the importance of accurately establishing the diagnosis.

Cadaveric investigations on 4200 spines demonstrated the incidence of spondylolysis as 4.2\%.\(^15\) The frequency of spondylolysis dramatically increases in young athletes. In a clinical study, 47\% of 100 young athletes who presented with low-back pain were diagnosed with spondylolysis.\(^11\) Jackson et al. demonstrated an 11\% incidence of spondylolysis among 100 female gymnasts.\(^8\) Although the diagnosis of spondylolysis at the initial stage is difficult with plain radiographs and CT, MRI has been recently shown to be effective.\(^2\),\(^18\),\(^23\) High signal changes (Modic signs) are seen in the ipsilateral pedicle in the early stage of a unilateral spondylolysis.\(^12\) Modic-type signal changes on spinal MRI were previously described in the endplates of the degenerated discs.\(^11\) These changes were anecdotally linked to the changes in the stress distribution within the disc and, thus, over the endplates. An FE analysis study also showed that the MRI signal changes in the pedicles in patients with spondylolysis could be associated with stress changes in the pedicle due to the redistribution of the loading after a fracture in the neural arch.\(^19\)

One of the limitations of this study is related to the application of loading. We simulated the extreme sagittal motion (extension) according to previous publications, which showed the contact of the tip of the superior facet pressing against the neighboring lamina in hyperextension. However, the magnitude of such loading was estimated according to the total load rather than being acquired from cadaver testing.

This study used an internal remodeling algorithm to observe the changes in the bone density for the simplicity of the problem. In other words, we did not allow our modeled pedicle to expand outward, which would simulate new bone formation at the cortex of the pedicle. A more sophisticated model might use this external remodeling algorithm, as well as internal remodeling to generate more clinically relevant results.

**Conclusions**

This study showed that unilateral spondylolysis might be related to sclerotic changes in the contralateral pedicle via a finite element model and remodeling algorithm. Our study showed increased stresses at the posterior vertebral structure and pedicles after a fracture at the pars. The remodeling algorithm resulted in an overall increase in density around the perimeter of the pedicle (cortex) similar to the clinical observations. The local bone loss in the cortical rim and trabecular core was observed, which might predispose the pedicle to development of microfractures.
Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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